The Role of Controlled Anticoagulation in Balloon Occluding Vertebral Arteries to Treat Giant Fusiform Aneurysms of the Basilar Artery

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Summary

We suggest and discuss the role of controlled anticoagulation therapy after the balloon occlusion of vertebral arteries to treat giant fusiform aneurysms in the basilar trunk.

Two cases of giant fusiform aneurysms were treated with balloon occlusion of vertebral arteries. Both of these patients suffered severe brain stem ischaemia. Anticoagulants were used to adjust the PTT to 1.5-2.5 times the normal level to control the formation speed of thrombosis inside the aneurysms.

Case 1 was obliged to suspend the anticoagulation therapy one week after occlusion because of digestive tract haemorrhage, and died of severe brain stem ischaemia. On autopsy, the sac of the aneurysm was totally occupied by the thrombus. Two perforating arteries feeding the brain stem arising from the wall of the aneurysm and infarction in the brain stem were found. Case 2 was anticoagulated strictly and progressively improved after three weeks. Anticoagulation was terminated after one month. Follow-up MRI showed the aneurysm had disappeared six months later.

Giant fusiform aneurysms in the basilar artery trunk can be treated with the balloon occlusion of vertebral arteries which induces thrombosis in the sac of aneurysm. Controlled anticoagulation should be given to slow down the thrombotic obliteration in the perforators arising from the aneurysm wall to the brain stem and give the brain stem have enough time to establish the sufficient collateral circulation.

Introduction

It is always a great challenge to treat intracranial no-neck giant fusiform aneurysms with a diameter larger than 2.5 cm in neurosurgery [1,2,3]. With the development of interventional neuroradiology, good results can be achieved by occlusion of the parent artery or embolisation of the aneurysm itself[5,6,8]. However it is still very difficult to treat giant fusiform aneurysms originating from the trunk of the basilar artery. This article is an in-depth comparative analysis of the treatment of two such aneurysms. It also discusses the architecture and treatment method on the basis of the autopsy result and suggests that controlled anticoagulation plays an important role after occluding the parent arteries of aneurysms.

Material and Methods

Case 1: A 12-year-old boy was admitted to our hospital with a four year history of headache and unsteady gait. Physical examination indicated paralysis of VII, IX XII cranial nerves and ataxia. CT and MRI showed a giant

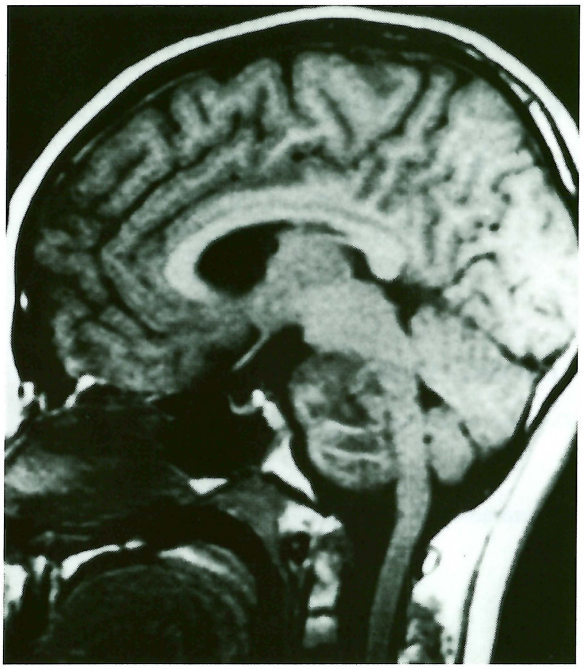

Figure 1 Case 1. MRI (T1 weighted) shows a giant mass in front of the brain stem.

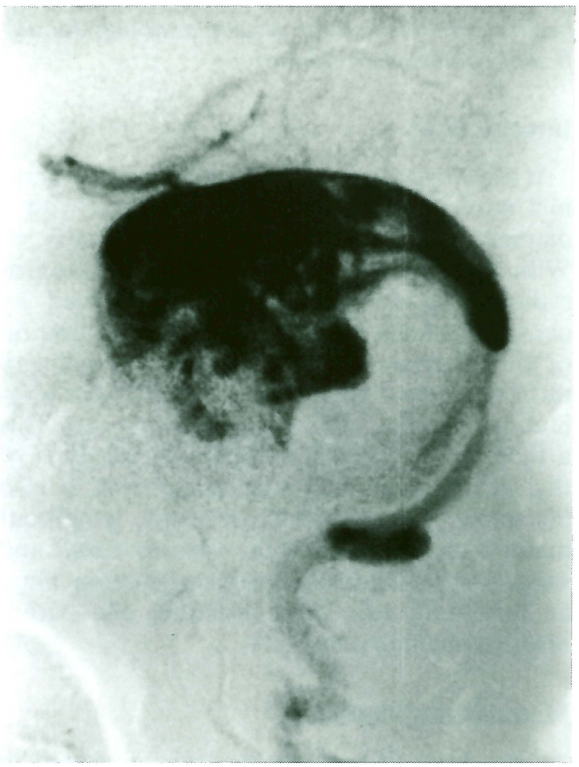

Figure 2 Case 1. Left vertebral angiography shows a giant aneurysm in the extreme dilatation of the basilar artery trunk, without a neck. Blood flow is turbulent. The aneurysm is partially thrombosed. SCA and PCA can be seen in the tip of the basilar artery.

spherical occupying mass in front of the brain stem (figure 1). Angiography under neuroanesthesia showed a giant fusiform aneurysm in the trunk of the basilar artery measuring 5x4x6 cm, which was fed by the left vertebral artery. There was a turbulent flow and partial thrombosis inside the aneurysm (figure 2). The right vertebral artery terminated above the posterior inferior cerebellar artery (PICA) and did not feed the aneurysm. The cerebellum and brain stem received their blood supply from the right PICA and both superior cerebellar arteries (SCAs). Both posterior communication arteries (PcomAs) were well-developed.

In the same session of angiography, a 30 minute occlusion test was performed with a n. 2 balloon attached to a Magic BD catheter (Balt Co. France) inflated in the intracranial segment of the left vertebral artery. The patient tolerated the test very well. Then the balloon was detached and another n. 1 balloon was placed below the first one to protect it. Post embolisation angiography showed a partial retrograde filling of the aneurysm from both PcomAs with slower blood flow. CT on the second day showed the aneurysm almost totally thrombosed and the size unchanged. Steroid and hypervolemic treatment were applied after occlusion without anticoagulation. Brain stem ischaemia symptoms such as central respiration failure, consciousness deterioration and right extremity plegia suddenly occurred 15 hours later. A respirator had to be used. Heparin and warfarin were applied 21 hours after embolisation to control PTT (Partial Thromboplastin Time) within 30 to 50 seconds. Heparin was stopped two days later and AT (Plasma Prothrombin Activity Test) was controlled within 20-40% only with warfarin.

The patient's condition improved slightly. Repeated Transcranial Doppler (TCD) showed a change in the direction, blood flow in the aneurysm slowing down gradually and vanishing eventually. Seven days later, severe haemorrhage of the digestive tract put an end to anticoagulation. The patient's condition deteriorated and he died 13 days later.

Autopsy demonstrated thrombosis inside the aneurysm, penetrating arteries feeding the brain stem arising from the aneurysm wall, some vessels thrombosed in the parenchyma, multifocal infarction in the brain stem (figure 3) and post-pneumonic consolidation.

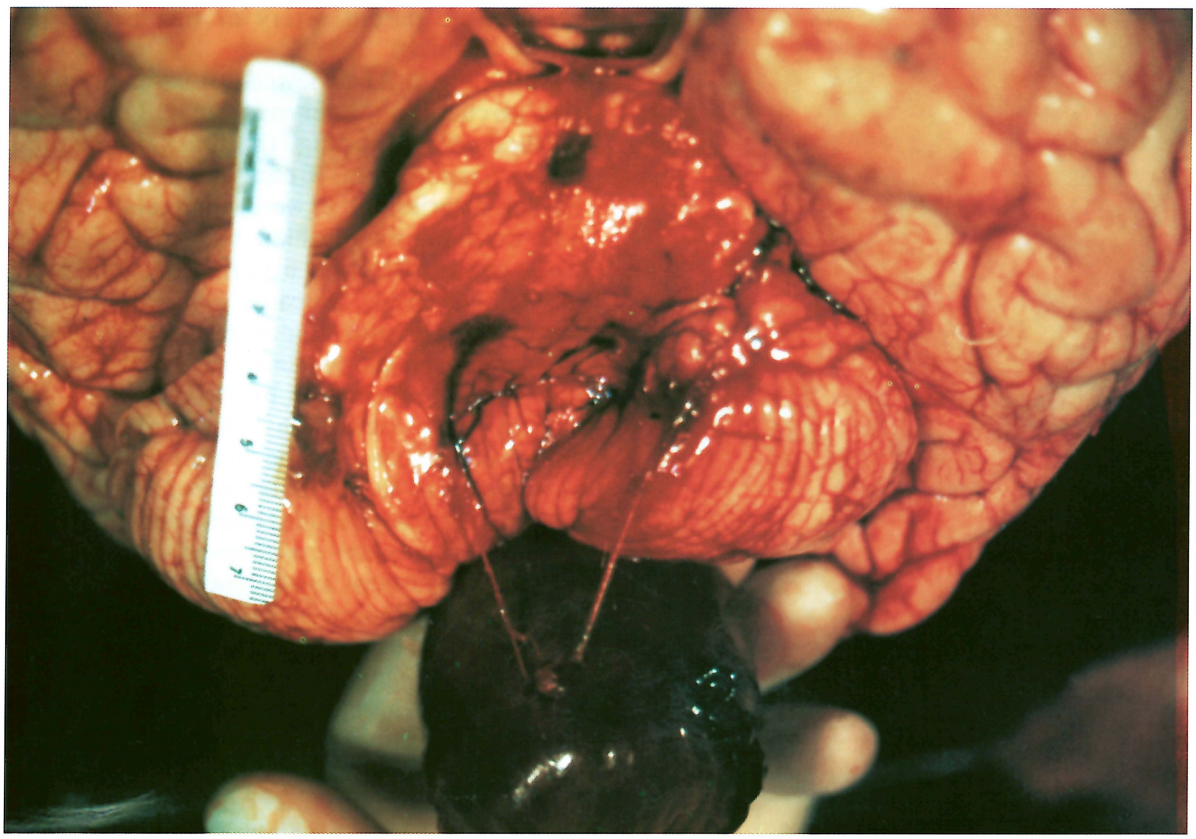

Figure 3 Case 1. Autopsy shows the whole trunk of the basilar artery extremely dilated into a giant aneurysm. Two small arteries feeding the brain stem are clearly demonstrated.

Case 2: A 10-year-old boy was admitted to hospital with a one year history of occasional headache accompanied by diplopia. Physical examination showed paralysis in the left IV, V, VI, VII and XII cranial nerves and ataxia. Head CT, MRI and angiography showed almost the same features as case 1 (figures 4, 5). The patient can also tolerated the 30 minute occlusion test very well.

Then the left vertebral artery was permanently occluded with a n. 1 balloon (Balt Co. France) detached in the intracranial segment. Heparin was continuously applied with a micropump to control PTT within 30-50 seconds after occlusion. Steroid and hypervolemic treatment were also given at the same time. CT on the second day showed most of the aneurysm thrombosed and the size of the aneurysm unchanged.

However, heparin had to be suspended twice at 36 hours and one week after occlusion respectively because of severe epistaxis and digestive tract haemorrhage. Symptoms of brain stem ischaemia such as unconsciousness and quadriplegia occurred just one hour after withdrawal of heparin. Heparin had to be given again with local haemostasis and the patient's condition improved.

Unfortunately, 11 days after the occlusion, platelet disastrously decreased due to the side effects of heparin and the haemorrhage in the digestive tract was almost out of control. Then heparin was replaced by low molecule heparin to continue the anticoagulation.

The symptoms of brain stem ischaemia finally stabilized and the patient's condition improved at the beginning of the third week after occlusion. Anticoagulation was completely suspended one month later and angiography showed no filling of the aneurysm. TCD showed a change in direction, blood flow in the aneurysm during this period slowing down and then vanishing. The boy fully recovered six months later and MRI showed the aneurysm

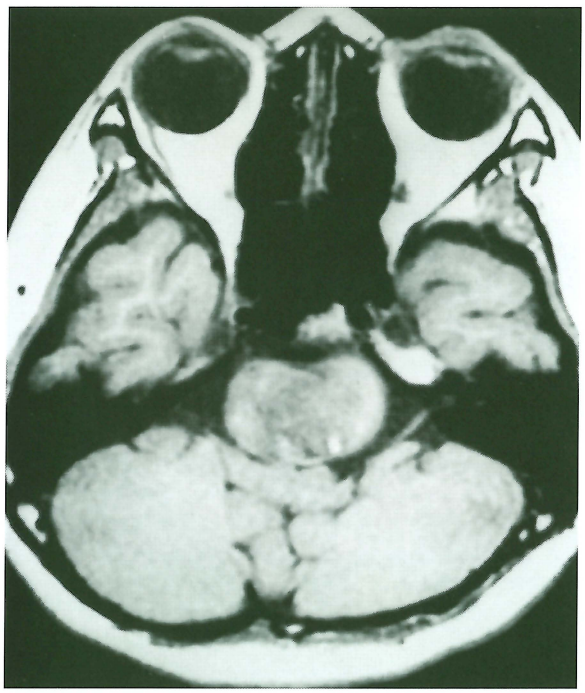

Figure 4 Case 2. MRI (T1 weighted) shows a giant mass in the front and the brain stem is severely compressed.

had completely disappeared (figure 6). In this case, the difference from the first one was that the anticoagulation was continued after occlusion, even when the haemorrhage complication occurred.

Discussion

Giant aneurysms in the basilar-vertebral artery are one of the most difficult cases in neurosurgery with an operative mortality as high as 50% reported in the literature [1,2]. Due to the facts that giant fusiform aneurysms in the trunk of the basilar artery have no neck and the normal perforators which feed the brain stem arise from the aneurysm wall, surgical clipping is extremely difficult with only a few cases reported to be excised and reconstructed [3].

Drake et Al first put forward the idea of surgical ligation or clipping of both vertebral arteries to reduce the direct impact of the blood flow on the aneurysm, resulting in thrombosis to treat basilar-vertebral aneurysms [4]. Detachable balloons were used to occlude the vertebral artery or basilar artery as an alternative to surgical ligation and clipping [5].

Other authors reported using GDC to occlude the parent artery or reconstruct the artery channel [6].

Sternberg et Al reviewed 201 cases of posterior circulation aneurysms treated with the method of occluding the basilar-vertebral artery. Among them, 46 cases are in the trunk of basilar arteries, 72% were completely cured, 4% were mildly disabled and 24% died. 36 cases of the 201 cases experienced brain stem ischaemia. These 36 cases were treated for hypervolemia and hypertension. When the ischaemia symptoms disappeared they were considered to be caused by blood flow malperfusion; when ischaemia symptoms persisted they were assumed to be caused by extensive thrombosis in the brain stem penetrating vessels from the thrombosed aneurysm [7].

These two giant fusiform aneurysms are extreme dilatations of the entire basilar arteries. Surgical clipping or excision is impossible. Coils cannot be held along the wall of the aneurysm to reconstruct the artery channel due to the turbulent blood flow in the aneurysm. Occlusion of the parent artery is the only solution.

After the bilateral vertebral arteries are occluded, the primary alternative blood supply to the brain stem comes from the PcomAs. When the PICA is below the balloon, collateral channels are thought to develop between the PICA and the AICA.

Some authors have investigated the relation between the diameter of the PcomA and prognosis after basilar-vertebral artery occlusion. The conclusion was that patients with a diameter of bilateral PcomAs larger than 1 mm are not likely to have ischaemia, while those with a diameter smaller than 1 mm on one side were more at risk for stroke, and those with less than 1 mm on both sides had the highest rate of ischaemia [7]. In both our cases, their bilateral PcomAs were larger than 1 mm in diameter, therefore, they both tolerated the 30 minute occlusion test well before the balloons were detached. The reliability of this occlusion test has been [5,7,8].

The two cases described in this article were given anticoagulation as soon as brain stem ischaemia symptoms appeared and as a result, they were markedly alleviated. But the symptoms deteriorated after anticoagulation was discontinued due to haemorrhage. Repeated CT scans were carried out after vertebral artery

occlusion showed a progressive thrombosis in the aneurysm after occlusion, but its size remained the same. The autopsy of case 1 showed penetrating vessels from the aneurysm wall to the brain stem and multifocal infarction in the brain stem caused by thrombosis in these vessels. So we believe the brain stem ischaemia of both cases was caused by extensive thrombosis of the brain stem feeding perforators arising form the the thrombosed aneurysm, but not caused by haemodynamic malperfusion or compression of thrombosed aneurysm.

Forsting et Al had also discovered the perforators to brain stem arising from the wall of a giant aneurysm in autopsy[9].

Previous literature mentioned that anticoagulation should be given when brain stem ischaemia occurs[7,9], but no reports have ever been made on specific application. This article suggests controlled anticoagulation to manage the formation speed of thrombosis inside the aneurysm to prevent fast thrombosis extending to normal vessels arising from the aneurysm wall, to gain enough time to let the collateral circulation gradually be established under the stimulation of slight ischaemia in the brain stem.

The anticoagulants used in our two cases were heparin, warfarin and low molecule heparin. We meticulously conducted a very fine regulation of dosage in line with the patient's symptoms, signs and laboratory examination results. We realize heparin produces a quick effect and is easy to control and monitor. It is better to use a micropump to maintain an appropriate blood concentration and control PTT at 1.5 to 2.5 times higher than the patient's basic level. But prolonged application of heparin may cause side-effects such as disastrous haemorrhage with a sharp decrease of platelets. Oral warfarin keeping AT between 30% to 50% also has same anticoagulation results but is inconvenient during the intensive care period. Low molecule heparin possesses the qualities of anticoagulation with a relatively lower haemorrhage risk and without the side-effect of reducing platelets. It can be used as an ideal medicine for long-term anticoagulation in critical patients. Thrombokinase (factor X) examination is only method of monitoring, which is difficult and costly. In the treatment of the second patient, the regulation of dosage relyied on the patient's clinical symptoms and

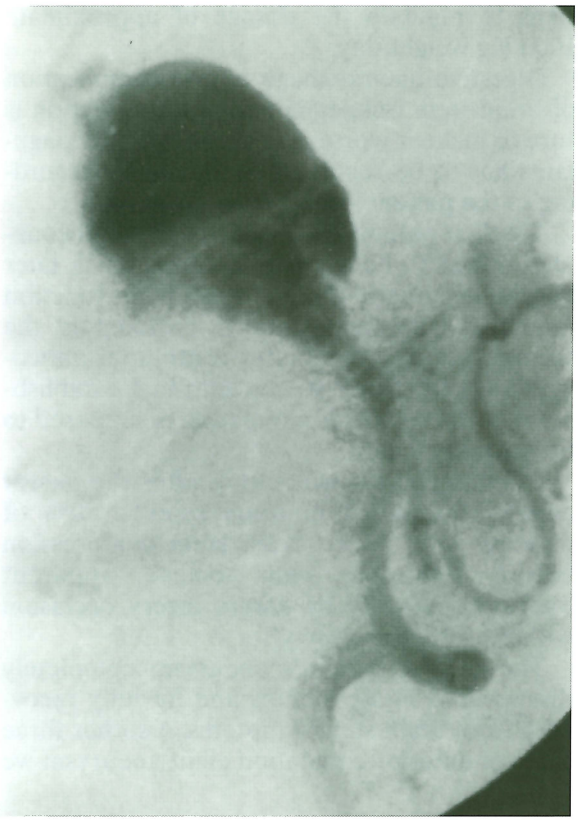

Figure 5 Case 2. Left vertebral angiography shows a giant aneurysm occupying most of the basilar artery trunk. SCA and PCA can be seen in the tip of the basilar artery.

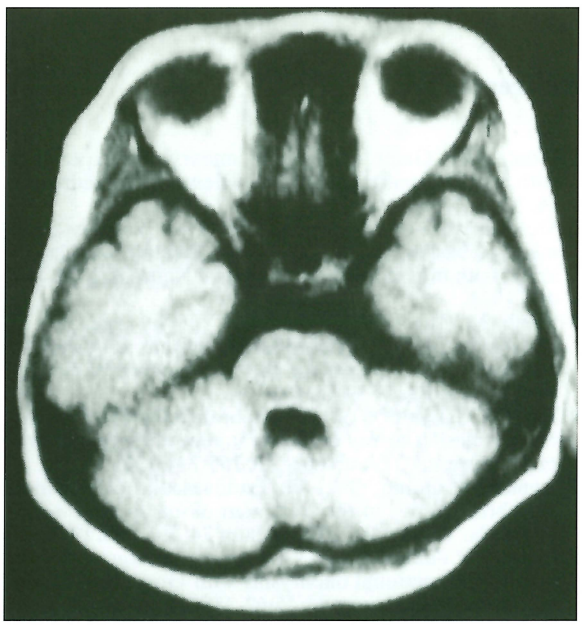

Figure 6 Case 2. MRI (T1 weighted) six month follow-up shows the mass in the front has disappeared and the brain stem has recovered its normal shape.

signs to regulate the dosage of approximate 100u/Kg weight/day.

Digestive haemorrhage is one complication of brain stem ischaemia and anticoagulation is sure to make it worse. The dosage of anticoagulants has to be regulated meticulously according to the patient's condition.

The first patient deteriorated after anticoagulation had to be discontinued one week later and he eventually died of brain stem function failure and its complications. Fortunately, the second case improved after continuous anticoagulation for three weeks. Gradual establishment of the collateral circulation is supposed to be the main factor.

Giant aneurysm will shrink after the parent artery is occluded in some reports. 80% of patients with severe brain stem compression before treatment can achieve apparent improvement only by parent artery occlusion without thromboectomy[9].

The second patient's aneurysm completely disappeared in six months and he fully recovered from brain stem symptoms. Another three cases of anterior circulation giant aneurysm we treated also led to aneurysms totally vanishing in about six months to one year after parent artery occlusion (to be published).

Conclusion

Balloon occlusion of the vertebral artery can induce thrombosis in the aneurysm with a change in blood flow direction to treat giant fusiform aneurysms in the trunk of the basilar artery. Some penetrating arteries feeding the brain stem arise from the aneurysm.

These perforators will be occluded in the process of thrombosis inside the aneurysm to cause brain stem ischaemia. The speed of thrombosis occlusion in these arteries can be slowed down with controlled anticoagulation and enough time can be gained for the establishment of collateral circulation of the brain stem to alleviate or prevent brain stem ischaemia.

Low molecular heparin may become an ideal medicine for long term controlled anticoagulation because of its reduced risk of haemorrhage and side-effects.

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